Streptococcus anginosus spondylodiscitis causing incapacitating back pain in an immunocompetent patient: A case report

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Abstract

Pyogenic spondylodiscitis is uncommon and usually presents in the setting of immunosuppression. *Streptococcus anginosus* group are opportunistic pathogens that rarely cause this infection. We present a case of an immunocompetent 45-year-old male with extreme lower back pain, not even relieved by opioids. A magnetic resonance imaging done the day before arrival showed multiple lumbar disk degeneration and lumbar spondylosis. Initial examinations did not show significant alteration. During inpatient admission, his values of erythrocyte sedimentation rate and C-reactive protein increased, and a new magnetic resonance imaging with contrast revealed signs of spondylodiscitis at the L2–L3 level. He underwent open surgery for tissue sampling and stabilization of the affected segment. Blood culture, disk sampling culture, and myeloculture were positive for *S. anginosus*. Additional examinations were negative for immunosuppression or any underlying condition, and the dental evaluation only showed mild gingivitis. The patient received intravenous antibiotics, and the pain significantly improved after surgery. He was finally discharged and completed 8 weeks of antibiotics. The erythrocyte sedimentation rate and C-reactive protein values were normal 6 weeks after surgery, and on a 1-year follow-up, the magnetic resonance imaging showed stable post-surgical changes with no signs of infection.

Keywords

Spondylodiscitis, Streptococcus anginosus, back pain

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Introduction

Spondylodiscitis is an uncommon musculoskeletal infection affecting the intervertebral disk and the adjacent vertebral body.¹ Whereas the most important pathogen is *Staphylococcus aureus*—responsible for more than half of the cases,² other bacteria can cause this infection in a smaller but increasing percentage, the *Streptococcus anginosus* group (SAG).^{1,3}

The SAG is a gram-positive bacteria that includes the *S. anginosus, Streptococcus constellatus*, and *Streptococcus intermedius* species.⁴ They are usually found in the oral cavity and are associated with dental and periodontal diseases.⁴ Many authors agree these bacteria are opportunistic pathogens since they leave their typical colonizing sites (digestive, respiratory, or genital tract) and enter the body's sterile zones.^{4,5}

Spondylodiscitis classically appears in patients with a decreased immune response or a preexisting local or

disseminated infectious disease.¹ Cases with none of these conditions have rarely been reported.^{2,3}

In this article, we present a case of an immunocompetent male patient with *S. anginosus* bacteremia and spondylodiscitis with no evident predisposing factor.

We obtained written informed consent from the patient for their anonymized information to publish this article.

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Case presentation

A 45-year-old man with a history of lumbar hernia L5–S1 arrived at the emergency room with a 7-day worsening back pain. The pain was non-radiating, shooting, and affected his ability to walk. One week ago, he increased his physical activity but did not experience any traumatic injury, fever, chills, or other symptoms. He self-medicated with over-the-counter painkillers and prednisone 75 mg daily for 7 days with no relief. A magnetic resonance imaging (MRI) performed the day before his arrival showed multiple disk degeneration (L2–L3, L4–L5, L5–S1) and lumbar spondylosis (L2–L3, L5–S1).

In the emergency department, his pain level was 8 out of 10 on the visual analog scale (VAS), he had mild tachycardia (100 beats per minute), and his vital signs were otherwise normal. Physical examination revealed facial expressions of discomfort, limited movement in his lower extremities due to pain, a positive Lasègue's⁶ test at 45° in the right lower limb, normal reflex testing (2+), and no motor or sensory deficits.

Initial laboratory evaluation showed hemoglobin 14.5 g/dL, white blood cell count 9730 cells/ μ l, platelet counts 260,000 per microliter, slightly increased ALT (45 IU/L), and normal AST (19 IU/L), alkaline phosphatase (100 IU/L), and creatinine (0.84 mg/dL).

During the first days after admission, the patient experienced severe pain even with intravenous (IV) analgesics. He initially received a continuous infusion of tramadol at 12.5 mg/h, plus acetaminophen 1g every 8h, ketoprofen 100 mg every 8h, and orphenadrine 60 mg every 12h. Tramadol was soon replaced by a continuous infusion of morphine at 40 mg/day (1.67 mg/h). Despite these medications, there was no significant improvement, and pain remained 8 to 10 out of 10 on the VAS, requiring IV morphine 5 mg as needed up to 3 times per day. The patient even avoided ambulation, defecation, or eating solid food, as minimal physical effort increased the pain.

Two days after admission, laboratory examinations (Table 1) showed rising values of C-reactive protein (CRP; 21.37 mg/dL) and erythrocyte sedimentation rate (ESR; 65 mm/h), raising suspicion of inflammatory spondyloar-thropathy. Further studies were performed, including *Brucella*, human immunodeficiency virus (HIV) test, QuantiFERON-TB test, and antinuclear antibodies, all of which were negative. We requested two blood culture tests and a new lumbar MRI with contrast. This imaging (Figure 1) revealed spondylodiscitis at the L2–L3 level and inflammation of the intervertebral disc, the adjacent bone marrow, and the surrounding tissue, causing moderate spinal canal stenosis. There were no abscesses or collections.

The interdisciplinary health team, consisting of internal medicine, infectious disease, neurology, neurosurgery, and interventional radiology doctors, discussed the case and reached a consensus on diagnosing pyogenic spondylodiscitis. They agreed to sample tissue for culture and biopsy and to start empiric antibiotic therapy immediately. Open surgery was preferred over the computerized tomography (CT)-scanguided method, as it would allow not only tissue sampling but also stabilization of the spine and pain management.

Open surgery of the L2–L3 segment with a posterior approach was performed. The procedure involved placing an awl on L2 and L3 pedicles and sampling bone marrow aspirate from the vertebral body for culture. Then, under fluoroscopy, bilateral instrumentation of L2 and L3 with transpedicular screws was performed. Under microscopy, right-sided L2 and L3 laminectomy and sampling of the yellow ligament and lamina were done, as well as L2–L3 microdiscectomy and disk sampling with local gentamicin application. Finally, titanium rods and plugs were placed, and saline irrigation was performed. Surgical findings included poorly vascularized subcutaneous cellular tissue, a hypervascularized disk with hard adjacent tissue adhered to the dural sac, and stenosis of the L2–L3 disk space, with no abscesses or collections.

Immediately after surgery, the patient started on IV ceftriaxone 1 g every 12 h plus vancomycin 1 g every 12 h. One of the two initial blood cultures tested positive for *S. anginosus* sp. This bacterium was also found in the L2–L3 disk sampling and myeloculture, and it was sensitive to penicillins and fluoroquinolones.

With these findings, the patient continued the established antibiotic therapy for 5 days. We performed additional examinations to look for the source of *S. anginosus* infection. Thoracic and abdominal CT scans were normal, and the transthoracic echocardiogram showed no signs of endocarditis. The dental evaluation revealed only gingivitis and no other issues. In addition, the ESR and CRP progressively decreased (Table 1).

The patient's pain improved significantly from the first postoperative days. We gradually reduced IV analgesics, and the patient increased mobilization and started ambulation. By the fifth postoperative day, the pain was minimal, and the patient did not need any additional painkillers. Due to the significant clinical improvement, the patient was discharged.

At home, he received IV ceftriaxone 1 g every 12 h and levofloxacin 500 mg every 12 h for 10 more days. On postoperative day 14, laboratory tests showed normal WBC (5130 cells/ μ L) and lower values of ESR (70 mm/h) and CRP (2.47 mg/dL). He completed ceftriaxone for 2 more weeks, which was then switched to oral cefuroxime 500 mg every 12 h for 6 weeks, plus oral levofloxacin 750 mg daily for 8 weeks. Six weeks after surgery, ESR (10 mm/h) and CRP (0.27 mg/dL) values became normal (Table 1). One year after surgery, a new lumbar MRI showed stable post-surgical changes at the L2–L3 level, with no inflammation or acute complications (Figure 2).

Discussion

We report an unusual case of an immunocompetent adult male with no predisposing factors for having *S. anginosus* spondylodiscitis.

Table I. Laboratory results t	hroughout the cours	e of the dise	ase.						
Variable	Reference range	On admission	2 days after admission	6 days after admission	10 days after admission/ postoperative day 3	Discharge/ postoperative day 5	2 weeks after surgery	4 weeks after surgery	6 weeks after surgery
Hemoglobin (g/dL)	13.5-17.5	14.5	4	13.8	12.8	12.2↓	12↓	12.6↓	
White blood cell count (/ μ L)	4500-11,000	9730	10,860	7850	5880	5970	5130	5560	
Platelet count (/µL)	150,000-475,000	260,000	273,000	301,000	388,000	494,000↑	534,000↑	348,000	
AST (IU/L)	0-40	61			24	24	29	81	
ALT (IU/L)	0-41	45↑			59↑	46↑	49↑	39	
Alkaline phosphatase (IU/L)	40-129	001			I80↑	I63↑	I33↑	119	
Albumin (g/dL)	3.5-5.2				4	4	4.2	4.2	
Total bilirubin (mg/dL)	0-1.1	0.32			0.38	0.24	0.11	0.27	
Direct bilirubin (mg/dL)	0-0.3	0.10			0.18	0.15	0.08	0.11	
Creatinine (mg/dL)	0.7-1.2	0.84	0.71		0.82	0.83	0.71		
C-reactive protein (mg/dL)	0-0.5		21.37↑	15.96↑	6.08 ↑	5.66↑	2.47↑	I.42↑	0.27
Procalcitonin (ng/mL)	0-0.5				0.1	0.09			
ESR (mm/h)	0-15		40↑	65↑	60↑	80↑	70↑	40↑	10

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AST: aspartate aminotransferase; ALT: alanine aminotransferase; ESR: erythrocyte sedimentation rate. \uparrow : High, \downarrow : Low



Figure 1. (a) Lumbar MRI, sagittal view at Stir sequence showed signs of spondylodiscitis at the L2–L3 level with inflammatory alteration of the intervertebral disc, the bone marrow adjacent to the vertebral platforms, causing moderate stenosis of the spinal canal caliber. No collections or abscesses were seen. (b) Axial view at T2-weighted sequence also revealed edema of surrounding soft tissue. MRI: magnetic resonance imaging.



Figure 2. (a) Lumbar MRI taken I year after surgery. The sagittal view at the T2-weighted sequence revealed straightening of physiological lumbosacral lordosis, without signs of listhesis, post-surgical alterations at the L2–L3 level, with no inflammatory changes at the surgical site. There was also mild spondylosis in the lumbar segment and signs of incipient degenerative disk disease. No herniated discs were observed. (b) Three-dimensional reconstruction of the lumbar spine showed loss of physiological lumbar lordosis, surgical changes at L2–L3 with no signs of acute complication, and L2–L3 intervertebral space narrowing with discrete sclerosis of the subcortical bone.

MRI: magnetic resonance imaging.

Pyogenic spondylodiscitis is an unusual condition that accounts for 2%–7% of all musculoskeletal infections.⁷ Previous studies reported an annual incidence as high as 5.8 per 100,000, being more prevalent in older people and men.⁸ Risk factors for having this infection include immunosuppression due to diabetes mellitus, chronic steroid

use, HIV, cancer, renal and liver failure, as well as advanced age, IV drug abuse, multimorbidity, rheumatic diseases, cardiovascular diseases, obesity, and previous spinal surgery.¹ *S. aureus* is responsible for more than 50% of the cases, whereas the *Streptococcus* group is found in 2%-11.6%.⁸

SAG spondylodiscitis is rarely reported,^{1–3,9–14} and most of these cases had at least one predisposing condition. For example, diabetes mellitus,^{1,12} alcohol abuse,¹ dental and periodontal disease,^{11,13} recent dental procedures,⁹ poor oral hygiene,¹ concomitant endocarditis,¹² spread of a contiguous infection,¹⁴ or obesity.¹⁰ Interestingly, a few patients also have no evident risk factors.^{2,3} From our knowledge, this is the first study in our country to report this condition.

Apart from chronic gingivitis, our patient had no other significant findings. Previous case reports showed periodontal disease increased the risk of spondylodiscitis,¹¹ and specifically gingivitis was associated with SAG abdominal abscesses¹⁵ and endocarditis.¹⁶ It raises the concern of whether this uncomplicated condition alone could be a risk factor for spondylodiscitis in an otherwise healthy man. Furthermore, this patient had recent use of oral glucocorticoids for 1 week, which started with the onset of symptoms. Although chronic steroid use is a known risk factor, in this case, the patient took them for a short time. However, it is unclear if this drug could have influenced the worsening of symptoms.

Early diagnosis is usually difficult since the symptoms are nonspecific, including new-onset or worsening back pain and fever. The increase in inflammatory markers such as ESR and CRP is also characteristic.¹⁷ These clinical and laboratory abnormalities require further workup, including blood cultures and imaging. Blood cultures are positive in 70% of cases of spondylodiscitis. Biopsy is recommended for all patients, mostly if blood cultures are negative,¹⁸ to confirm the diagnosis, to distinguish between pyogenic or granulomatous, and to rule out malignancy.¹⁷ A spine MRI is the imaging test of choice and has high sensibility and specificity, 96% and 93%, respectively.^{1,17}

Our patient did not have a fever which is present in less than 50% of the cases,¹⁹ but he received acetaminophen for pain management, which could also control the temperature rise. The severe back pain, the increased CRP and ESR levels, and the findings in the second MRI made the pyogenic cause very likely, which was finally demonstrated by the positive cultures.

Treatment should focus on infection control, pain management, and preserving spine functionality.¹ Conservative management with IV antibiotics is the first choice for noncritically ill patients and initiates after obtaining cultures. However, empiric antimicrobial therapy should start immediately in sepsis, hemodynamic instability, neutropenia, or neurologic compromise.¹⁰

The initial antibiotic regimens should cover *S. aureus* (including the methicillin-resistant staphylococci aureus), streptococci, and gram-negative bacilli, such as the combination of vancomycin plus a third- or fourth-generation cephalosporin,¹⁸ and may change with culture results. Antibiotics should last 6–12 weeks, and switching to oral regimens depends on clinical and inflammatory markers

improvement.^{17,18} Our patient started empirical antibiotics after tissue sampling, and then he received targeted antibiotics for *S. anginosus*, with a total duration of 10 weeks and normalization of ESR and CRP values on week 6.

Surgical management is reserved for cases of severe pain, epidural abscess, compression of spinal cord or nerve roots, and failure to respond to conservative treatment.¹⁰ It includes debridement of infected tissue, drainage, antibiotic irrigation, decompression, and, in some cases, spine stabilization.²⁰ Open surgery can have an anterior, posterior, or combined approach. Whereas the anterior approach has been the traditional technique, it may result in longer operation duration and higher complication rates.²¹ By contrast, the posterior approach has demonstrated adequate results with fewer adverse effects.²¹

Spine stabilization is crucial in spondylodiscitis treatment, and instrumented surgery is critical to achieve this. There are concerns about whether using external devices such as screws increases the risk of biofilm formation and recurrent infection. However, research shows that spinal instrumentation does not lead to higher reinfection rates than non-instrumented surgery.²² By contrast, decompression and debridement alone result in a higher reoperation rate since they do not provide stabilization.²⁰ In our case, the patient underwent surgical management to obtain samples for biopsy and cultures and to alleviate pain with laminectomy, discectomy, and spine stabilization using screws. One year after surgery, the patient experienced no pain and had a stable spine with no signs of infection recurrence.

Conclusion

S. anginosus spondylodiscitis is an uncommon pathology that affects mainly immunocompromised patients. Rarely, it can occur in patients with no identifiable risk factors. Pain refractory to adequate analgesic therapy plus the increase in ESR and CRP should raise suspicion of this diagnosis and require further examinations, including imaging and cultures. Antibiotic treatment is the first management option. It should immediately start after obtaining cultures, whereas surgical management is reserved for those cases refractory to conservative management, with epidural abscess or severe pain.

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Author contributions

M.F.T.-N.: Conceptualization, investigation, data curation, writing—original draft. L.A.: Conceptualization, investigation, data curation, supervision, writing—review and editing. G.V.: Conceptualization, supervision, writing—review and editing.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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References

- Potsios C, Xaplanteri P, Zoitopoulos V, et al. Pyogenic spondylodiscitis due to *Streptococcus constellatus* in an immunocompromised male patient: a case report and review of the literature. *Case Rep Infect Dis* 2019; 2019: 1–4.
- Gangone R, Findlay I, Lakkireddi PR, et al. A very rare spontaneous group-C streptococcal constellatus spondylodiscitis: a case report. *J Orthop* 2009; 6(3): e7.
- Lim SW, Lim HY, Kannaiah T, et al. *Streptococcus constellatus* spondylodiscitis in a teenager: a case report. *Malays* Orthop J 2017; 11(3): 50.
- Pilarczyk-Zurek M, Sitkiewicz I and Koziel J. The clinical view on *Streptococcus anginosus* group—opportunistic pathogens coming out of hiding. *Front Microbiol* 2022; 13: 956677.
- Jiang S, Li M, Fu T, et al. Clinical characteristics of infections caused by *Streptococcus anginosus* group. *Sci Rep* 2020; 10(1): 9032.
- Das MJ and Nadi M. *Lasegue sign*. Treasure Island, FL: StatPearls Publishing, 2024.
- Cheung DA, Langshaw A and Rivera-Rivera E. Cryptosporidium diagnosed on endoscopic biopsy in a paediatric patient with inflammatory bowel disease. *Case Rep* 2018; 2018: bcr-2017-222015.
- Kehrer M, Pedersen C, Jensen TG, et al. Increasing incidence of pyogenic spondylodiscitis: a 14-year population-based study. *J Infect* 2014; 68(4): 313–320.

- Ramhmdani S and Bydon A. *Streptococcus intermedius*: an unusual cause of spinal epidural abscess. *J Spine Surg* 2017; 3(2): 243.
- Jin Y and Yin X. Acute pyogenic spondylitis caused by *Streptococcus constellatus* in an obese patient: a case report. *Infect Drug Resist* 2022; 15: 4361–4367.
- Koruga N, Rončević A, Soldo Koruga A, et al. Aggressive pyogenic spondylitis caused by *S. constellatus*: a case report. *Diagnostics (Basel)* 2022; 12: 2686.
- Roig-Marín N, Roig-Rico P, Calbo-Maiques J, et al. Triad of endocarditis, spondylodiscitis and epidural abscess due to *S. intermedius. Med Interna México* 2022; 38(6): 1296–1299.
- Quast MB, Carr CM and Hooten WM. Multilevel lumbar spine infection due to poor dentition in an immunocompetent adult: a case report. *J Med Case Reports* 2017; 11(1): 328.
- Manasrah N, Nanja Reddy S, Al Sbihi A, et al. *Streptococcus intermedius*: unusual presentation and complication of lung abscess. *BMJ Case Rep* 2021; 14(11): e245675.
- Terzi HA, Demiray T, Koroglu M, et al. Intra-abdominal abscess and primary peritonitis caused by *Streptococcus anginosus. Jundishapur J Microbiol* 2016; 9(6): e33863.
- 16. Yoshino Y, Kimura Y, Sakai T, et al. Infective endocarditis due to a rare pathogen, *Streptococcus constellatus*, in a patient with gingivitis: a case report and review of the literature. *Cent Eur J Med* 2013; 8(4): 489–492.
- Guerado E and Cerván AM. Surgical treatment of spondylodiscitis. An update. *Int Orthop* 2012; 36(2): 413–420.
- Berbari EF, Kanj SS, Kowalski TJ, et al. 2015 Infectious Diseases Society of America (IDSA) clinical practice guidelines for the diagnosis and treatment of native vertebral osteomyelitis in adults. *Clin Infect Dis* 2015; 61(6): e26–e46.
- Gouliouris T, Aliyu SH and Brown NM. Spondylodiscitis: update on diagnosis and management. *J Antimicrob Chemother* 2010; 65(Suppl 3): iii11–iii24.
- Lee JJ, Sadrameli SS, Sulhan S, et al. The role of instrumentation in the surgical treatment of spondylodiscitis and spinal epidural abscess: a single-center retrospective cohort study. *Int J Spine Surg* 2022; 16(1): 61–70.
- Zhang HQ, Wang YX, Feng GC, et al. Posterior-only debridement, bone fusion, single-segment versus short-segment instrumentation for mono-segmental lumbar or lumbosacral pyogenic vertebral osteomyelitis: minimum five year followup outcomes. *J Orthop Surg* 2022; 17(1): 388.
- Pluemer J, Freyvert Y, Pratt N, et al. An assessment of the safety of surgery and hardware placement in de-novo spinal infections. A systematic review and meta-analysis of the literature. *Glob Spine J* 2023; 13(5): 1418–1428.